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EXAMINER

FOSTER, CHRISTINE E

ART UNIT	PAPER NUMBER
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1641

NOTIFICATION DATE	DELIVERY MODE
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ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

poreilly@licataandtyrrell.com

Office Action Summary	Application No.	Applicant(s)	
	10/511,719	KIM ET AL.	
	Examiner	Art Unit	
	Christine Foster	1641	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 20 February 2007 and 18 May 2007.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 33-44 is/are pending in the application.
- 4a) Of the above claim(s) 39 and 44 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 33-38 and 40-43 is/are rejected.
- 7) ☒ Claim(s) 33, 40 is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☒ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☒ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413) |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | Paper No(s)/Mail Date. _____ |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

Amendment Entry

1. Applicant's amendments, filed 2/20/07 and 5/18/07, are acknowledged and have been entered. Claims 17-32 were canceled. New claims 33-44 were added.
2. The newly presented claims are subject to the election of species set forth in the restriction requirement mailed 7/14/06. In Applicant's Reply of 8/11/06, the species of SEQ ID NO:3 was elected as the species of recombinant β ig-h3 protein. Newly submitted claims 39 and 44, which recite similar subject matter as previous claims 24 and 32, do not read on this elected species for similar reasons as set forth in the previous Office action at pages 2-3, item 3. Accordingly, claims 39 and 44 are hereby withdrawn from consideration as being directed to non-elected species. Claims 37-38 and 42-43 are being examined in light of the elected species of SEQ ID NO:3. See 37 CFR 1.142(b) and MPEP § 821.03.
3. Accordingly, claims 33-44 are currently pending, with claims 33-38 and 40-43 subject to examination below.

Objections/Rejections Withdrawn

4. The objection to the specification is withdrawn in response to Applicant's amendments thereto and in light of the sequence listing filed on 5/18/07.
5. The objections to and rejections of claims 17-21, 23, 25-29, and 32 are moot in light of the claims' cancellation.

Claim Objections

6. Claims 33 and 40 objected to because of the following informalities:
7. Part (b) of claim 33 indicates that the abbreviation " β ig-h3" stands for "Transforming growth factor induced gene-h3". However, the specification discloses the full term as transforming growth factor- β induced gene-h3" (see page 2, line 23). Appropriate correction is required.
8. Claim 40 recites a recombinant protein and "an antibody thereof". Applicant is requested to employ more precise language to clarify the relationship between the antibody and the protein.

Claim Rejections - 35 USC § 112

9. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.
10. Claims 36-38 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

New Matter

11. New claim 36 recites step (a) in which a recombinant β ig-h3 or β ig-h3 fas-l domain protein is reacted with the urine sample, followed by step (b) in which the mixture is contacted with a matrix that is also coated with the recombinant protein and then contacted with a

secondary antibody in step (c). Such a method notably fails to invoke the use of a primary antibody directed against the antigen β ig-h3, and apparently employs only a recombinant protein and a secondary antibody to detect antigen. The method also involves two separate steps in which the sample is contacted with recombinant protein. Applicant's Reply of 2/20/07 states that no new matter has been added but does not particularly point out where support may be found for the amendments to claim 36.

Original claim 5 recited step (1) in which the sample was reacted with *antibody* against the protein. The specification discloses only competition assays in which the sample is reacted with an anti- β ig-h3 antibody and then subsequently added to a matrix having a coating of a recombinant β ig-h3 protein as a control (see especially page 18, line 18 to page 19, line 5). The originally disclosed methods differ from those instantly claimed in that they involve the use of a primary anti- β ig-h3 antibody and do not involve multiple steps in which the sample is contacted twice with a recombinant β ig-h3 or β ig-h3 fas-l domain protein, both in solution and then as a coating on a solid phase matrix.

Support could not be found for a method as now claimed in which recombinant protein is first reacted with the sample, reacted again with the recombinant protein coated on a matrix, and then with a secondary antibody. One skilled in the art would not envisage possession of such methods, which involve only use of a recombinant β ig-h3 and a secondary antibody, because it is unclear how β ig-h3 protein could be detected without the use of a primary anti- β ig-h3 antibody.

Enablement

12. Claims 33-38 and 40-43 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The claims as currently amended are drawn to methods and kits for diagnosing damage to the kidneys at an early stage, based on measurement of β ig-h3 in urine samples.

In Example 4, the specification discloses the results of an experiment in which β ig-h3 levels were measured in subjects with type II diabetes, including those diabetic subjects who also had renal disease (as indicated by microalbuminuria, overt proteinuria, or outright chronic renal failure): Applicants found that in all type II diabetes populations, urinary β ig-h3 levels were increased as compared to normal controls (Table I).

However, it can be seen in Table 1 that β ig-h3 levels were increased even in those diabetic subjects *without* renal disease (Table 1, "Type II DM population"), which appears to represent a false positive finding. In particular, while the data in Table 1 show a distinction between β ig-h3 levels in normal subjects as compared to type II diabetic subjects (those with and without renal disease), the distinction between β ig-h3 levels in diabetic subjects without renal disease (the second table entry) and those with renal disease (the third, fourth and fifth table entries) is less apparent, being within the reported margins of error. In other words, while the data show a clear separation between β ig-h3 levels in type II diabetes vs. normal subjects, it would appear that with respect to the populations of diabetic vs. diabetic with renal disease that

the separation is not statistically significant. For example, compare “Type II DM” (without renal disease) β ig-h3 levels of 101.9 with “Type II DM + overt proteinuria” (with renal disease) β ig-h3 levels of 105.4. These levels are the same within the margins of error reported. There is no statistically significant elevation in β ig-h3 levels between the disease and control populations.

These data indicate that β ig-h3 is specifically elevated in Type II diabetes, but do not necessarily indicate that β ig-h3 is a marker that is specific to kidney damage, since it was apparently elevated in all diabetic subjects regardless of whether they had kidney damage or not.

Applicants discuss these experiments on p. 38, line 14 to p. 39, line 5, but draw a different conclusion, stating that the data of Table 1 would indicate that β ig-h3 is an *early* marker of renal disease since it is detectable even in the absence of any clinical symptoms of renal disease (such as microalbuminuria or proteinuria).

It is known that not all subjects with type II diabetes would also have kidney disease. For example, the American Diabetes Association reports that about 20-30% of patients with diabetes develop evidence of nephropathy (kidney disease) (“Nephropathy in Diabetes”; Diabetes Care Vol. 27, Supplement 1 (2004), pages S79-S83; especially at page S79, left column). Therefore, only about 20-30% of the patients in the “Type II DM” population of Table 1 would develop kidney disease. However, in the instant specification, only the β ig-h3 levels for the entire population are reported; no attempt is made to break down the population in terms of those diabetic subjects who later developed signs of kidney damage and those who did not. As such, it is not known whether only those subjects in the “Type II DM” population who had early kidney damage displayed elevated β ig-h3 levels, or alternatively whether all subjects had elevated levels

(in which case elevated β ig-h3 would not be useful as an early stage kidney damage marker).

There is insufficient data presented to conclude that β ig-h3 can be used to diagnose kidney damage at an early stage of disease.

The conclusion that β ig-h3 is an early marker of kidney damage since it was elevated in diabetic subjects without other signs of kidney that β ig-h3 levels cannot be accepted without question based on the data reported. One skilled in the art would recognize that when investigating a new candidate biomarker for validity in diagnosis of disease, the finding of that biomarker in the *absence* of disease would normally be considered to be a false positive until this could be ruled out by further experimentation. See LaBaer et al. (of record) at Figure 1 in particular. LaBaer et al. further teach that for biomarkers to be used for diagnosis, quantitative values must be established that set the boundary between a positive and negative test (see p. 105, "Disease Diagnosis").

The specification fails to disclose or recite any such "cutoff" values with respect to diagnosis of kidney damage. Based on the data reported, the skilled artisan would face an undue burden of examination in selecting such a cutoff value, since β ig-h3 levels were *the same* within the reported margins of error among diabetic subjects with and without signs of kidney damage. For example, one might select a β ig-h3 cutoff level of 105.4 ± 14.9 (based on the β ig-h3 levels observed for diabetics with kidney damage as indicated by proteinuria). However, this is the same level (within the margins of error reported) that would also be expected for diabetics with no signs of kidney damage (101.9 ± 17.1), and 70-80% of such patients would *not* subsequently develop disease. The specification fails to teach the skilled artisan how to diagnose early kidney damage since it fails to teach how early kidney damage can be discriminated from diabetes.

Furthermore, as discussed above, the specification also notably fails to provide any retrospective data in which the diabetic subjects without any overt clinical symptoms were followed over time for subsequent development of kidney damage, and in which β ig-h3 levels were assessed in terms of those subjects who went on to develop kidney disease and those who did not.

It is noted that MPEP 2164.03 teaches that “the amount of guidance or direction needed to enable the invention is inversely related to the amount of knowledge in the state of the art as well as the predictability of the art. In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970). The amount of guidance or direction refers to that information in the application, as originally filed, that teaches exactly how to make or use the invention. The more that is known in the prior art about the nature of the invention, how to make, and how to use the invention, and the more predictable the art is, the less information needs to explicitly stated in the specification. In contrast, if little is known in the prior art about the nature of the invention and the art is unpredictable, the specification would need more detail as how to make and use the invention in order to be enabling.”

In the instant case, little is apparently known about the use of urinary β ig-h3 for early stage diagnosis of kidney damage. However, the data fail to include appropriate controls that would rule out false positive findings. Levels of β ig-h3 were examined only in subjects who were also known to have diabetes, and elevations were seen for all diabetic subjects with and without signs of kidney disease. It is therefore not clear that β ig-h3 is specifically elevated in kidney damage, as opposed to being generally elevated in diabetes.

Consequently, the data presented in the specification would at best support the use of urinary β ig-h3 as a marker for *diabetes*, but do not reasonably enable the skilled artisan to use β ig-h3 as a marker of early kidney damage, since β ig-h3 was elevated to a similar degree in all diabetic subjects regardless of whether kidney damage was present or not. The data do not support a statistically significant difference in disease vs. control subjects, such that the skilled artisan would face an undue burden in using β ig-h3 as a marker to diagnose early stage kidney damage.

13. The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

14. Claims 33-38 and 40-43 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

15. Claim 33 recites a method for diagnosing damage to kidneys "**at an early stage**". Similarly, claim 40 refers to a kit for diagnosing damage "at an early stage". The term "early" is a relative term which renders the claim indefinite. The term "early" is not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. Because the specification does not define what time periods would be encompassed by the term "early", one skilled in the art would not know whether a method, performed on a patient at a certain point in time, would fall within the scope of the claim or not.

16. Claim 36 recites the limitation “the reactant” in parts (b) and (c). There is insufficient antecedent basis for this limitation in the claims since there is no prior mention that any components are reacting.

17. Claims 36-38 are rejected under 35 U.S.C. 112, second paragraph, as being incomplete for omitting essential elements, such omission amounting to a gap between the elements. See MPEP § 2172.01. The omitted elements are: **a primary anti- β ig-h3 antibody**. Claim 36 depends from claim 33, which requires that the β ig-h3 protein is measured by antigen-antibody reaction. However, claim 36 only recites a recombinant protein that is contacted with the sample (step (a)); the recombinant protein coated onto a matrix (step (b)), and a secondary antibody (step (c)). The claim fails to recite a primary anti-antigen antibody (i.e., a primary anti- β ig-h3 antibody), which would be essential for antigen-antibody reaction to occur as recited.

Claim Rejections - 35 USC § 103

18. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

19. Claims 40-43 are rejected under 35 U.S.C. 103(a) as being unpatentable Harlow & Lane (Antibodies: A Laboratory Manual (1988) Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY, pages 558-559, 570-576, 586-589 and 591-593) in view of Gilbert et al. (“Renal expression of transforming growth factor- β -inducible gene-h3 (β ig-h3) in normal and diabetic

rats” *Kidney International* **54** (1998), 1052-1062) and Zuk et al. (US 4,208,479), or in the alternative as being unpatentable over Harlow & Lane in view of Gilbert et al., Zuk et al., and Ratti et al. (US 5,629,167).

Harlow & Lane teach various assay formats for detecting and quantitating antigens, including antigen competition assays, which employ a sample of pure or nearly pure antigen as a standard, which is mixed with a test sample containing an unknown amount of the antigen to be detected (see especially p. 559, “Detecting and quantitating Antigens”, and p. 570). The standard antigen and the antigen in the test sample compete for binding to a ligand (antibody) that is specific for the antigen. The reference also teaches that samples containing known amounts of pure antigen can also be used to generate standard curves, which can be used to make assays quantitative (p. 576).

The reference teaches detection of antigens in general using a sample of the antigen and an antibody therefor by competitive assay, but does not teach detection of β ig-h3.

Gilbert et al. teach that the expression of β ig-h3 is significantly increased in the kidneys of rats with experimentally-induced diabetes (see the entire document, especially the abstract; the paragraph bridging p. 1052-1053; p. 1056-1057, “Discussion”; and p. 1059-1060. Gilbert et al. teach that β ig-h3 levels are correlated with those of TGF- β , which is known to play a pathogenetic role in diabetic kidney disease, and further that β ig-h3 may be useful as an index of TGF- β 1 bioactivity in the kidney (p. 105).

Zuk et al. teach kits, in which reagents used in performing assays are combined together for convenience and for enhancing accuracy (column 22, lines 20-53).

Therefore, it would have been obvious to one of ordinary skill in the art to employ the competition assay format of Harlow & Lane, which involves providing a sample of the protein to be detected, in order to detect β ig-h3 because Gilbert et al. teach that this protein is an index of TGF- β 1 bioactivity in the kidney. It would have been further obvious to package all of the reagents necessary (i.e., sample of the β ig-h3 protein and an antibody therefor) for performing such an assay into a kit as taught by Zuk et al. for convenience.

With respect to the limitation that the protein is “recombinant”, Applicant is reminded that the patentability of a product does not depend on its method of production (MPEP 2113). In the instant case, no differences in structure are clearly implied as a result of the recombinant production of the protein. Therefore, it is Applicant’s burden to establish an unobvious difference between the claimed product and the prior art product.

However, even if the process by which the protein is produced is given weight, the claimed invention is nonetheless found obvious over Harlow & Lane in view of Gilbert et al., Zuk et al., and Ratti et al. for the following reasons.

Although Harlow & Lane do not specifically teach that the antigen is produced by recombinant DNA technology, such technology as well as its advantages were well known in the art. For example, Ratti et al. teach a significant advantage of producing protein by recombinant DNA techniques rather than by isolating and purifying a protein from natural sources is that equivalent quantities of the protein can be produced by using less starting material than would be required for isolating the protein from a natural source. Therefore, it would have been further obvious to prepare the protein antigen by recombinant techniques to obtain larger quantities of protein.

With respect to claim 41, Harlow & Lane teach the buffer/washing solution PBS (e.g. p. 586), secondary antibody (see p. 574-575), chromogenic substrates (p. 592), and stop solution (H₂SO₄, p. 593).

With respect to claim 42, although Gilbert et al. teach measurement of β ig-h3 in an experimental rat model of diabetes, it would have been further obvious to employ recombinant human β ig-h3 (i.e., SEQ ID NO:3) an antibody specific therefor in order to extend the laboratory model experiments to the study of human disease.

With respect to claim 43, it is noted that the claim employs open transitional language in reference to the recited sequences (“comprises”). As Harlow & Lane teach use of purified antigen to be detected in competition with the same antigen that is detected, and because Gilbert et al. teach the full-length β ig-h3 protein (see p. 1056), the purified full-length β ig-h3 would necessarily comprise the 4th fas-l domain that is a portion of the protein.

With respect to the intended use of the kit as recited in the preamble, it is noted that the recited purpose does not result in a structural difference in the kit reagents and accordingly, has not been construed as a claim limitation. See MPEP 2111.02. Therefore, although Gilbert et al. is silent as to measurement of β ig-h3 in relation to “early” kidney damage, the kit of Harlow & Lane, Gilbert et al. and Zuk et al. is structurally indistinguishable from that claimed and therefore reads on the instant product claims since the kit would be capable of performing the recited intended use.

Response to Arguments

20. Applicant's arguments with respect to now-canceled claims 17-23 and 25-31 have been considered and are technically moot in view of the new ground(s) of rejection. However, certain of the arguments will be addressed below as they bear on the rejections set forth above.

21. With respect to the rejections of claims 17-23 and 25-31 under § 112, first paragraph for lack of enablement, Applicant argues that the results in Table 1 do support the use of use of β ig-h3 in relation to diagnosis of "early" kidney damage that cannot be diagnosed by conventional detection methods (Reply, pages 13-14). However, it is maintained for reasons of record that the studies reported in Table 1 have not ruled out false positives. In particular, Table 1 only reports only aggregate data for the entire population of diabetes subjects with no symptoms of kidney disease. It is not disclosed whether levels were altered in those subjects who subsequently developed symptomatic kidney disease vs. those who were not. Since β ig-h3 was elevated in all diabetic subjects (with and without signs of kidney disease), the data fail to support the use β ig-h3 as a marker of early kidney disease (as opposed to being a marker of diabetes).

Furthermore, Applicant is claiming methods of detecting early stage kidney damage in any type of subject. Arguments directed towards latent diabetic renal disease (see pages 14-15) are therefore not on point since they are not commensurate with the scope of the claims.

22. With respect to the rejections of claims 26-31 under § 103(a) as being unpatentable over Harlow & Lane in view of Gilbert et al. and Zuk et al., although it is acknowledged that the teachings of Gilbert et al. do not fairly teach diagnosis of early kidney damage as now claimed, the references have now been applied in the rejection of kit claims 40-43.

Applicant's arguments that the references do not teach or suggest detection of β ig-h3 in urine as an indicator of early kidney damage (Reply, page 19) are not persuasive in relation to the product claims, since a recitation of the intended use of the claimed invention must result in a structural difference between the claimed invention and the prior art in order to patentably distinguish the claimed invention from the prior art. If the prior art structure is capable of performing the intended use, then it meets the claim. In the instant case, it is maintained for reasons of record that the teachings of Harlow & Lane, Gilbert et al., and Zuk et al. render obvious the assembly of β ig-h3 and an antibody specific therefor into kit form. Since these are the same reagents as claimed instantly, they would also be capable of performing the recited intended use.

Conclusion

23. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event,


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however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Christine Foster whose telephone number is (571) 272-8786. The examiner can normally be reached on M-F 8:30-5. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Long Le can be reached at (571) 272-0823. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.


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